Surgical Considerations of Dissecting Aneurysm of the Aorta[*]

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THE WELL-DEFINED CLINICAL and pathological features of dissecting aneurysms of the aorta have been recognized for over a century, but treatment for this grave disease has remained largely symptomatic and unsatisfactory.[13, 17, 21, 26, 27] Unlike sacciform and fusiform aneurysms in which excisional therapy may be readily employed, dissecting aneurysms of the aorta constitute an entirely different problem, owing to the pathologic nature of the lesion. For one thing it is rarely sufficiently well localized, the lesion often extending throughout the extent of the aorta, to permit application of this form of surgical attack. For another it usually arises in the upper reaches of the aorta about the aortic arch, and most often in the ascending aorta[26] (Fig. 1). It is thus apparent that the surgical approach to this problem requires somewhat different principles than those underlying excisional therapy.

The challenging nature of this disease is further emphasized by its highly fatal course. This is well illustrated by the reports of a number of observers. Shennan,[26] for example, in his analysis of 143 collected cases found that death occurred within 24 hours in 58 per cent and in one day to one week in 26 per cent. Among the cases recorded by Weiss, Kinney and Mahr,[29] only about 10 per cent survived for a significant period of time. More recently Levinson,

Edmeades and Griffith[14] reported that 36 per cent of their patients died within 48 hours of onset, 37 per cent within three to 60 days, and only 25 per cent with the chronic or so-called healed form of the disease survived three months to eight years. Thus, it would appear that the condition is rapidly fatal in from 75 to 90 per cent of the cases.

Few attempts have been made to attack this problem by surgical means, and all have proved unsuccessful. The first of these is believed to be the report of Gurin, Bulmer and Derby,[9] in 1935, whose efforts were directed primarily toward relief of acute arterial insufficiency in the right lower extremity of a patient with an acute dissecting aneurysm of the aorta. After exploring the femoral artery and finding it pulseless but intact, the right external iliac artery was exposed about three inches above Poupart's ligament and found to have "an infiltration of dark blood in its lateral third, extending as far as could be seen in both directions," representing the dissecting aneurysm. No expansile pulsation was present in any part of the vessel. After applying occluding clamps to the artery above and below, a longitudinal incision about 1 inch in length was made in the unaffected anterior wall. Upon entering the true lumen of the artery it was found narrowed by the dissecting aneurysm, and completely occluded by an atheromatous mass. The intima and media opposite the atheroma were incised from within the vessel, thus creating an opening into the false lumen of the dissecting aneurysm,

EXTENT OF DISSECTION 287 CASES (SHENNAN)

ascending aorta
84 (29.9%)

transverse
arch 6(2%)

ascending aorta
to mid arch
11 (3.9%)

ascending aorta
to distal arch
31 (10.8%)

ascending aorta to
descending thoracic
21 (7.3%)

ascending aorta
into abdominal aorta
30 (10%)

left subclavian
to diaphragm
10 (3.4%)

ascending aorta to iliacs
or beyond 41 (14.2%)

left subclavian to abdominal
aorta or beyond 53 (18.4%)
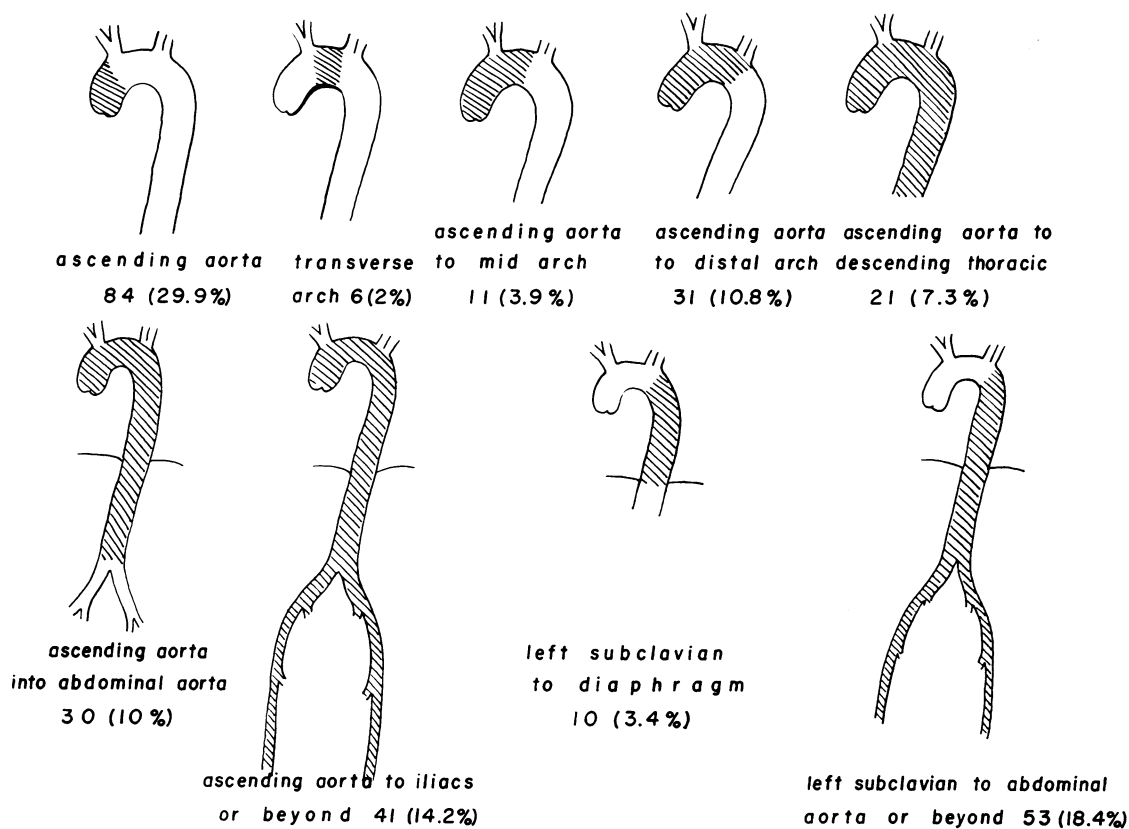
FIG. 1. Diagram showing results of analysis of 243 collected cases of dissecting aneurysms of aorta (Shennan[26]).

through which bright arterial blood spurted upon momentary release of the proximal clamp. Following closure of the incision in the anterior wall of the vessel and removal of the occluding clamps, pulsations were restored in the extremity, but the patient died on the sixth postoperative day of renal failure. In 1953, Johns[12] reported the repair by suture closure of a perforated dissecting aneurysm of the abdominal aorta, but this patient also succumbed from renal insufficiency on the eighth postoperative day. The pathologic features of this case would suggest that it was a rather unusual and perhaps atypical form of dissecting aneurysm.

In a more typical case of an acute dissecting aneurysm Shaw[25] has recently re-

ported application of a surgical procedure somewhat similar to that employed by Gurin and associates. Here again the major manifestations were those of acute arterial insufficiency of the lower extremities. Upon exposure transperitoneally of the abdominal aorta and iliac arteries no pulsations were palpable in these vessels. The procedure employed consisted essentially in making a longitudinal incision into the aneurysm in the lower abdominal aorta, extracting a soft clot from its lumen, permitting free bleeding from above, making a small window into the true aortic lumen by excising a 2×2.5 cm. piece of the internal aneurysmal wall, closure of the lower plane of the dissecting aneurysm with interrupted mattress sutures, and repair of the aortot-
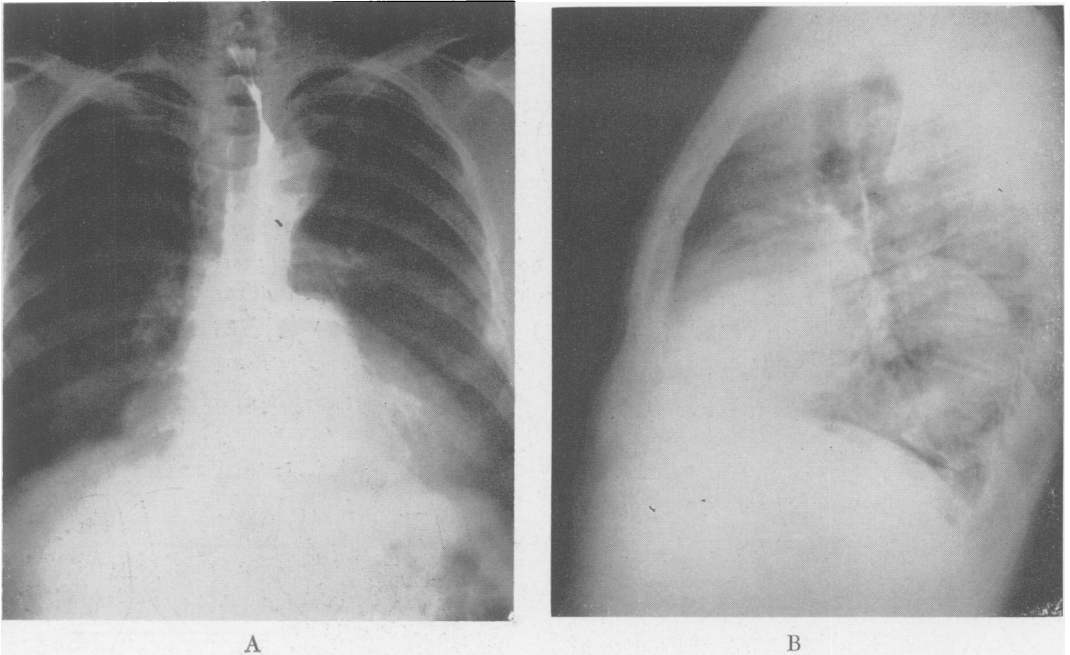
FIG. 2 A and B. Roentgenograms of chest in Case 1 in postero-anterior and lateral projections, showing tortuous, dilated descending thoracic aorta extending into left paravertebral sulcus.
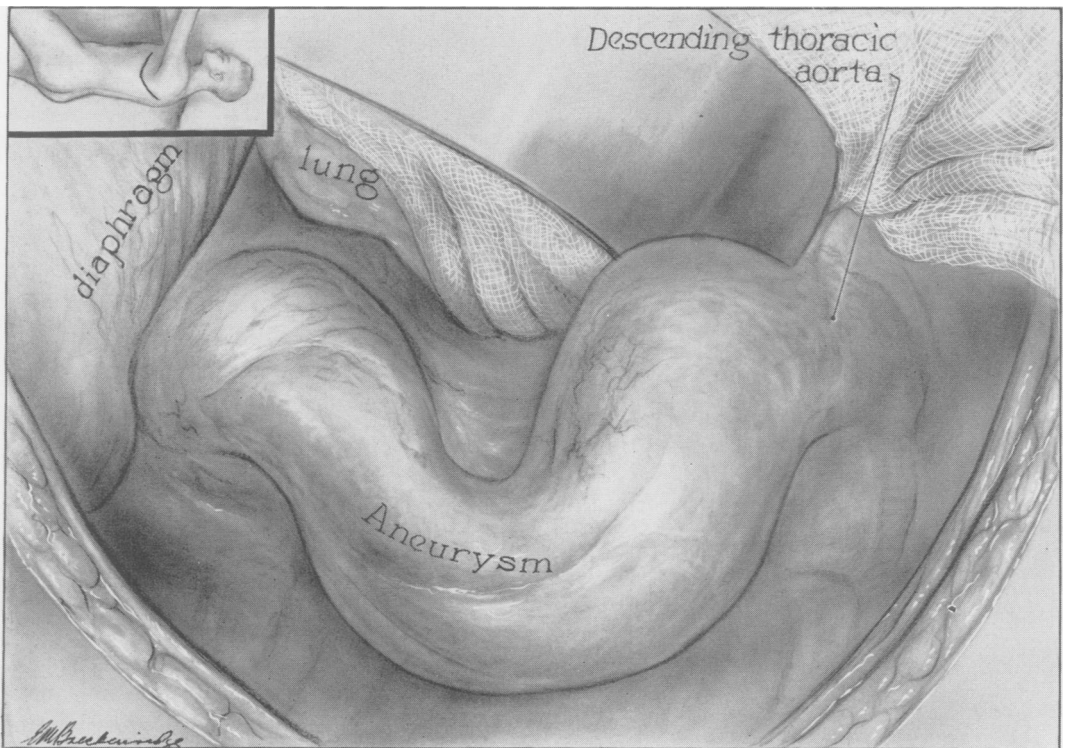

FIG. 3. Drawing made at operation in Case 1, showing aneurysm projecting posteriorly and to the left.

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Fig. 4. Photograph made at operation in Case 1, showing aneurysm mobilized prior to application of occluding clamps.

omy. Following completion of the procedure good pulsations were restored in the aorta, iliac, femoral and popliteal arteries. Unfortunately the patient died of renal insufficiency on the ninth postoperative day.

Our experience with the surgical treatment of this disease is based upon six cases. Although the underlying principles of the surgical procedures employed were essentially similar, certain variations in their application seemed desirable on the basis of differences in the pathologic features of the lesions encountered (Fig. 1).

REPORT OF CASES

Case 1. P.M., 58-year-old white man, was admitted to the Veterans Administration Hospital on May 4, 1954, complaining of shortness of breath on exertion and swelling of the feet of 3 months'

duration. One year previously he had experienced sudden, severe, constricting pain in the chest, which began posteriorly and radiated along the rib margins bilaterally. He was hospitalized for about 10 days, and during the first 3 or 4 days the pain was of such severity that opiates gave only partial relief. After release from that hospital he was well until 3 months prior to the present admission, when exertional and paroxysmal nocturnal dyspnea developed and the feet began to swell.

Physical examination revealed a blood pressure of 230/130 mm. Hg. Minimal excursion of the diaphragm was noted, and generalized expiratory wheezes were heard throughout both lung fields. The heart was enlarged to the left, and there were both apical and aortic systolic murmurs. A loud, prolonged systolic murmur was audible over the posterior aspect of the left side of the chest beneath the scapula.

Laboratory studies on admission were not remarkable, and serologic test for syphilis was negative. Radiologic examination of the chest revealed a
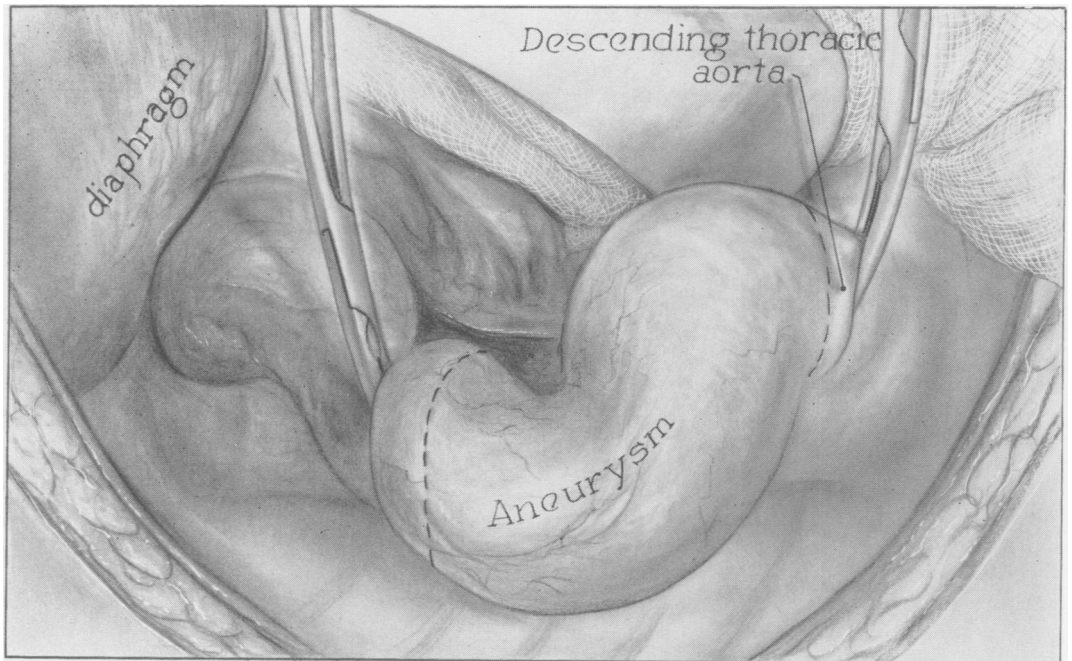
FIG. 5. Drawing made at operation in Case 1, showing occluding clamps applied above and below the aneurysm and the levels of transection of the aorta.
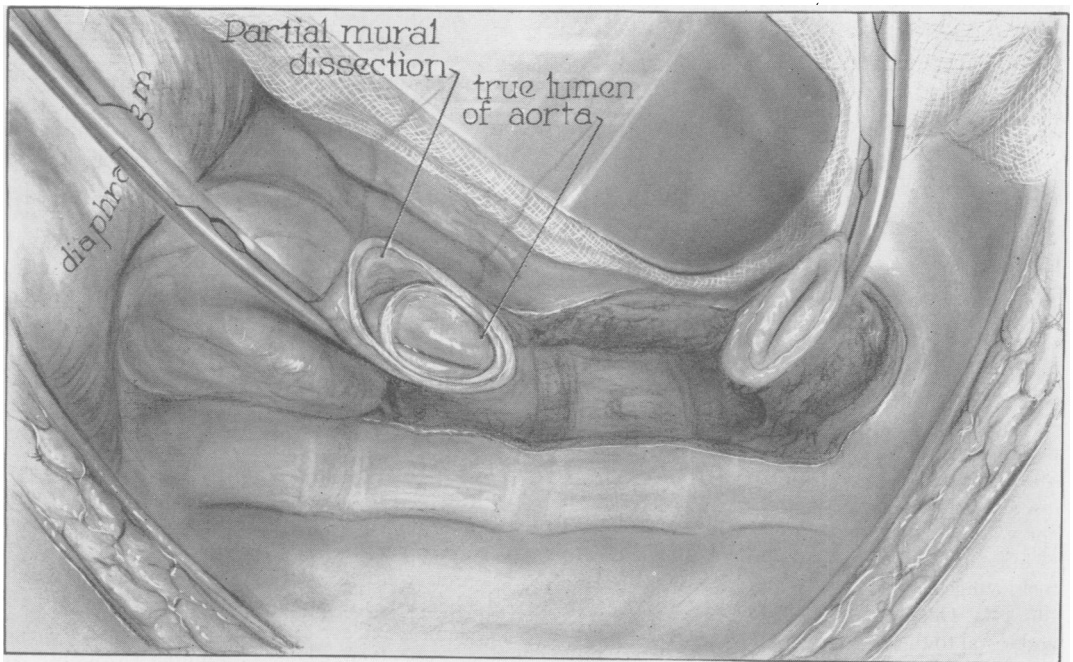

FIG. 6. Drawing made at operation in Case 1, showing double lumen in distal segment of arota and single lumen proximally.

rounded density in the posterior inferior mediastinum that projected into the left hemithorax, and appeared to be a markedly tortuous, dilated, descending thoracic aorta (Fig. 2 A and B). An electrocardiogram showed evidence suggestive of a previous posterior myocardial infarction.
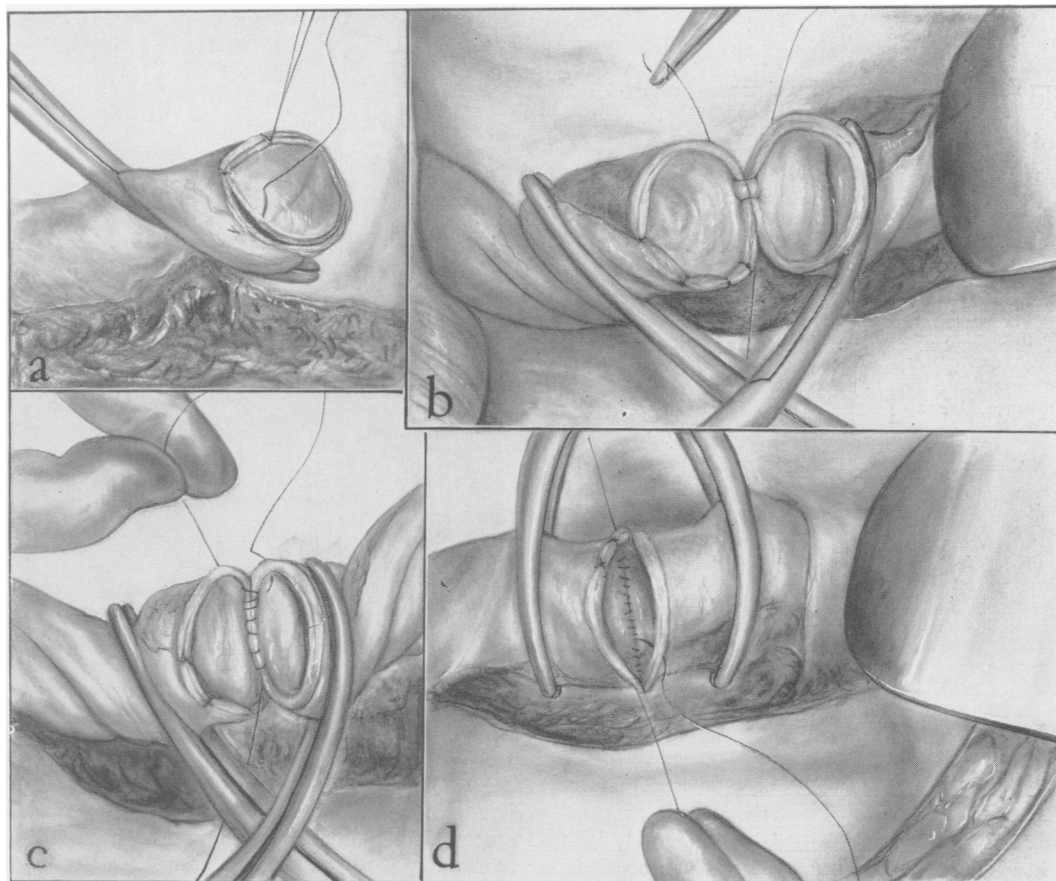
FIG. 7. Drawing made at operation in Case 1, showing (A) obliteration of the false lumen in the distal segment; and (B, C and D) anastomosis of the two ends by continuous suture.

On July 7, 1954, operation was performed under induced body hypothermia after lowering rectal temperature to 90° F. Thoracotomy was performed through a left posterolateral incision, with resection of the fifth rib. The lung was retracted anteriorly, revealing a U-shaped aneurysm of the descending thoracic aorta which projected posteriorly and to the left (Fig. 3). The mediastinal pleura was incised, and umbilical tapes were passed about the aorta above and below the aneurysm. Three pairs of intercostal arteries were ligated and divided, thus mobilizing the aneurysm throughout its length (Fig. 4). Occluding clamps were applied proximal and distal to the aneurysm, and the intervening segment of aorta was excised (Fig. 5). Examination of the distal end of the aorta revealed a double lumen, formed by the dissecting process (Fig. 6). The false lumen was obliterated by re-approximating the dissected layer with several interrupted sutures (Fig. 7 A). Restoration of aortic continuity

was then performed by end-to-end anastomosis (Figs. 7 B, C, D, and 8). The occluding clamps were removed 44 minutes after application.

Examination of the resected specimen revealed a well-formed thrombus in the false lumen. The proximal level of aortic resection was above the aneurysm, and the area of original intimal tear was demonstrated (Fig. 9).

Convalescence was uneventful. Three months later roentgenograms, including angiocardiogram, revealed a relatively normal outline of the descending thoracic aorta (Fig 10 A and B).

Case 2. W.C., a 46-year-old white man, was admitted to the Methodist Hospital on November 20, 1954, with a diagnosis of aneurysm of the thoracic aorta. The patient had been in good health until May, 1954, whe he developed severe pain in the left chest, at which time a roentgenogram disclosed an aneurysm involving the distal arch and de-
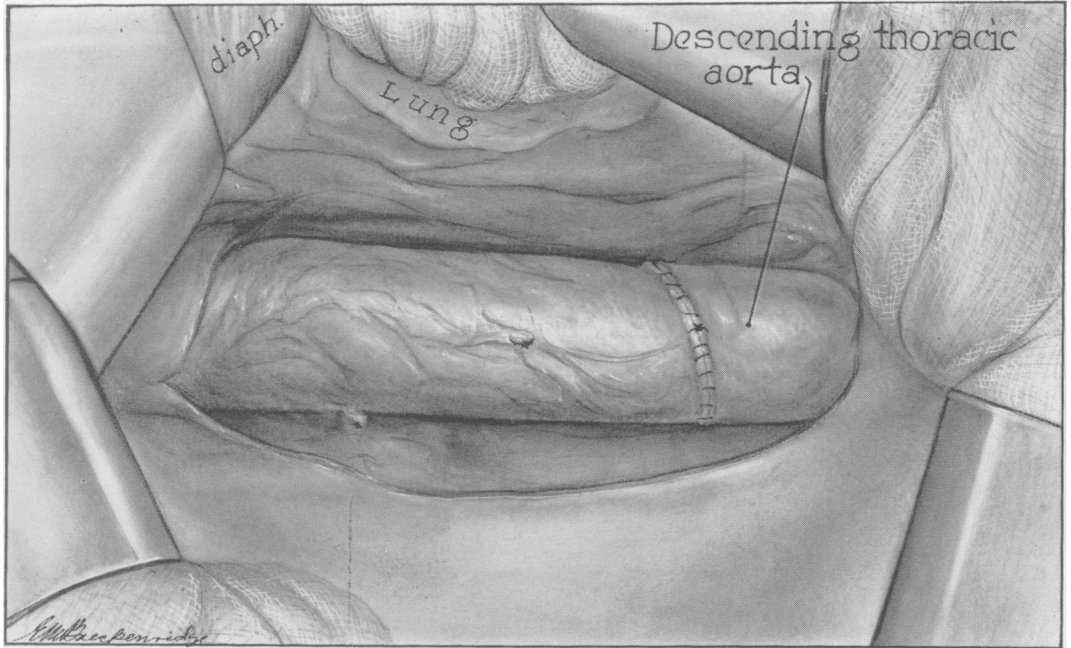
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FIG. 8. Drawing made at operation in Case 1, showing completed anastomosis.

scending thoracic aorta. Convalescence from this illness was satisfactory, and the patient remained relatively symptom-free until November 11, 1954, when he experienced hemoptysis, which continued intermittently for the next 6 days.

Physical examination revealed a well-developed, well-nourished white man. The blood pressure was 170/112 mm. Hg. A Horner's syndrome was evident on the left. There were prominent pulsations in the suprasternal notch, and a soft systolic murmur was audible over the aortic valvular zone of the precordium. Pulsations in the lower extremities were normal.

Roentgenographic examination of the chest revealed a fusiform aneurysm of the thoracic aorta which began in the posterior portion of the arch and involved the upper portion of the descending thoracic aorta (Fig. 11 A and B). It appeared that the aneurysm had increased in size significantly since its detection 5 months previously. An angiocardiogram outlined the aneurysm, and demonstrated the false passage created by the dissecting process (Fig. 12). There appeared to be an anomalous right subclavian artery arising from the posterior aspect of the distal arch and passing behind the esophagus. The left subclavian artery was not visualized.

Laboratory findings revealed a hemoglobin of 11.3 Gm., and hematocrit, 36 per cent. Urinalysis showed an occasional erythrocyte and hyaline cast. The electrocardiogram indicated left ventricular preponderance, but no evidence of myocardial infarction.

On November 23, 1954, operation was performed under general body hypothermia, at rectal temperature of 83° F. Left thoracotomy was performed through a posterolateral incision, resecting the fifth rib. Exploration revealed an aneurysm of the distal arch of the aorta, beginning at the origin of the anomalous right subclavian artery and extending to the middle third of the descending thoracic aorta (Table I). The right and left common carotid arteries arose from a common trunk near the center of the aortic arch. The right subclavian artery was ligated and divided. Since the left subclavian artery arose from the aneurysm, it, too, was ligated and divided near its origin. Occluding clamps were applied just distal to the common carotid artery above and to the descending thoracic aorta below, and the aneurysm was excised. Examination of the specimen disclosed a dissecting aneurysm, with the false passage beginning just below the proximal line of resection and extending below the level of transection distally (Fig. 13). At the lower level of aortic transection there was a double lumen, and therefore the false passage was obliterated suturing the dissected layers together. Aortic continuity was then re-established by insertion of a
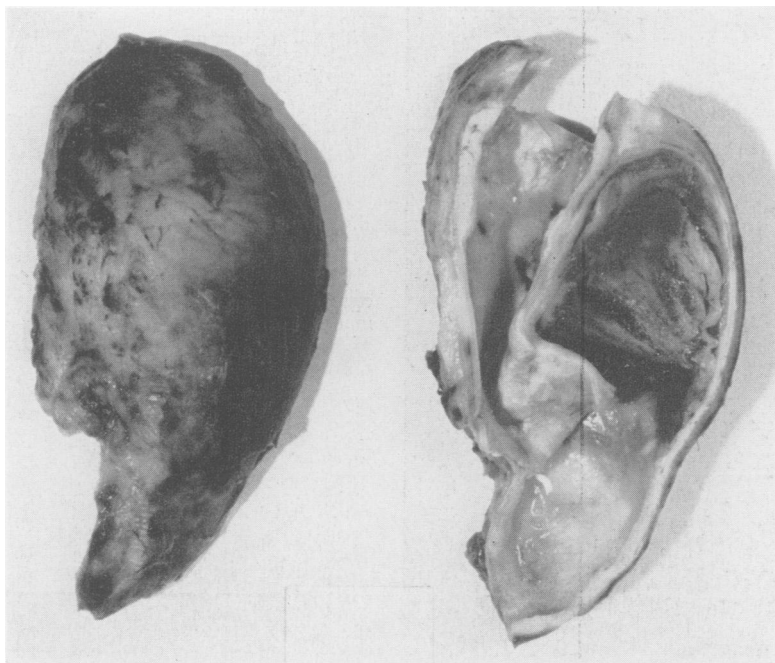
FIG. 9. Photograph of specimen removed in Case 1, showing well formed thrombus in the false lumen.

freeze-dried homograft. The duration of aortic occlusion was 65 minutes. During the rewarming period, respiration was being assisted when a left pneumothorax developed in spite of the underwater drainage with suction. Cardiac arrest occurred rapidly, followed by ventricular fibrillation, but cardiac resuscitation, including cardiac massage and electrical defibrillation, restored heartbeat. The patient did not regain consciousness, however, and death occurred 14 hours after operation.

Autopsy revealed the presence of a dissecting aneurysm, which extended from the descending thoracic aorta into the common iliac arteries (Fig. 14). No re-entry point was found. The homograft was intact.

Case 3. C.F., a 40-year-old white woman, was admitted to Methodist Hospital on January 6, 1955, for treatment of an aneurysm of the arch of the aorta. In September 1953 a severe *pectus excavatum* was repaired elsewhere, and after operation the left lung failed to expand. A diagnosis of stenosis of the left main stem bronchus was made at bronchoscopy. Atelectasis subsequently failed to respond to repeated bronchoscopy, so in December, 1953, a left thoracotomy was performed. At that time an aneurysm of the arch of the aorta just distal to the left subclavian artery was discovered. Because the aneurysm was pressing on the bronchus, a left

pneumonectomy was done, but no attempt was made to resect the aneurysm. After this operation she had been well until one month prior to the present admission, when pain developed in the left interscapular region and left shoulder.

Physical examination revealed a blood pressure of 115/80 mm. Hg. The chest was somewhat asymmetrical due to the previous repair of the *pectus excavatum* and left pneumonectomy, but no abnormal masses or pulsations were evident. The remainder of the physical examination was essentially normal.

Roentgenograms disclosed opacification of the entire left hemithorax, and displacement of the heart to the left. Angiocardiograms revealed a large dissecting aneurysm of the thoracic aorta, beginning just distal to the left subclavian artery and extending distally to the level of the diaphragm (Fig. 15). The true aortic lumen was heavily opacified, and appeared relatively normal in diameter. An electrocardiographic tracing was normal, and laboratory studies revealed no significant findings.

On January 10, 1955, left thoracotomy was performed through the bed of the resected fifth rib. The pleural cavity contained a small quantity of serous fluid. There was a globular aneurysm, measuring approximately 12 cm. in its greatest diameter in the distal arch of the aorta, beginning just distal to the left subclavian artery and extending to the
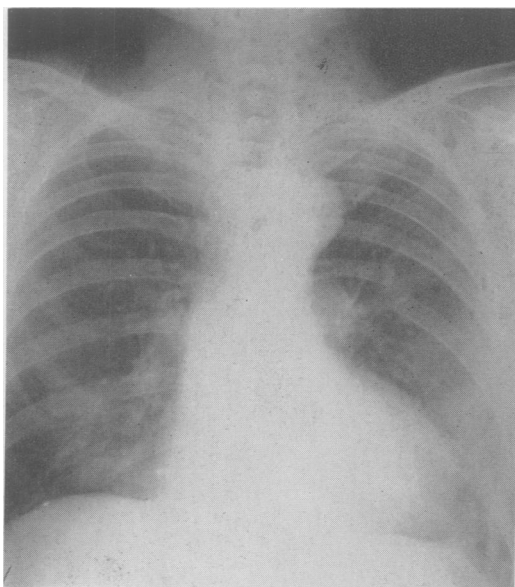
A

FIG. 10. A (top) and B (bottom). Roentgenogram of chest and angiocardiogram in Case 1 made 3 months after operation, showing absence of the aneurysm and relatively normal outline of descending thoracic aorta.
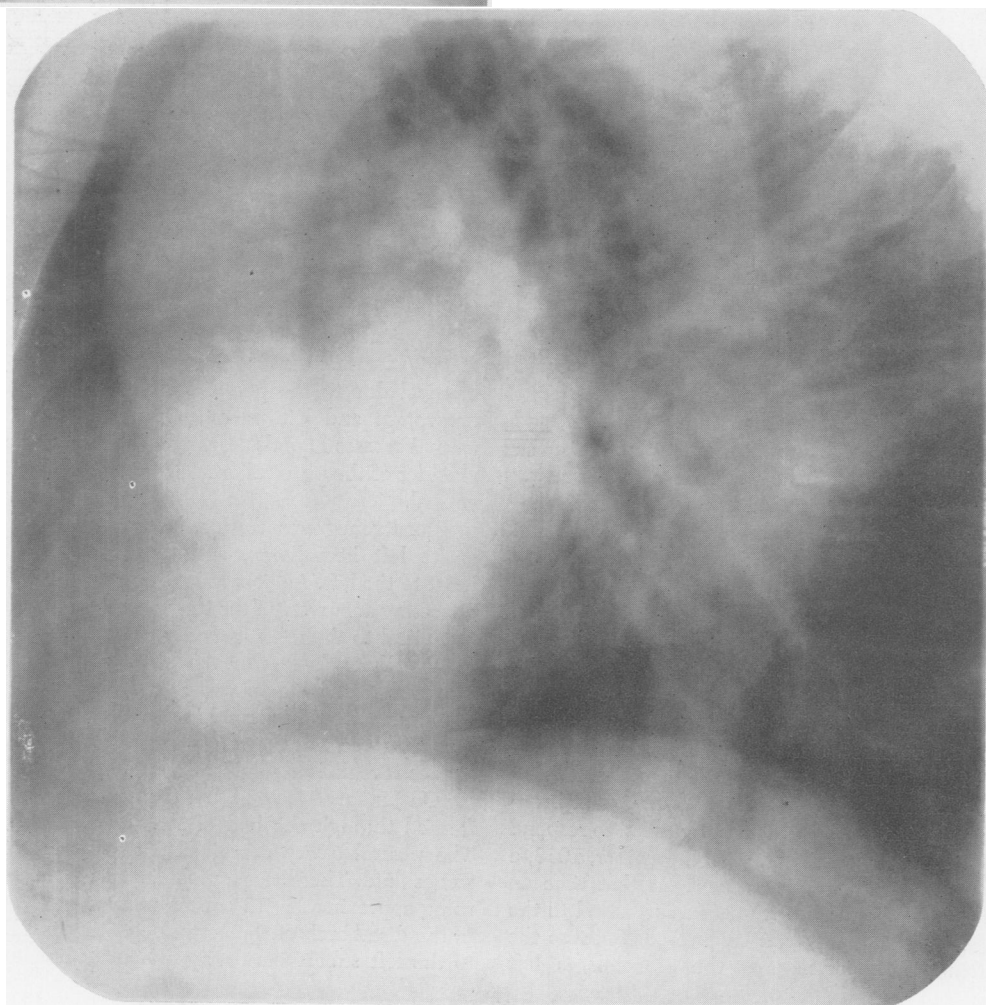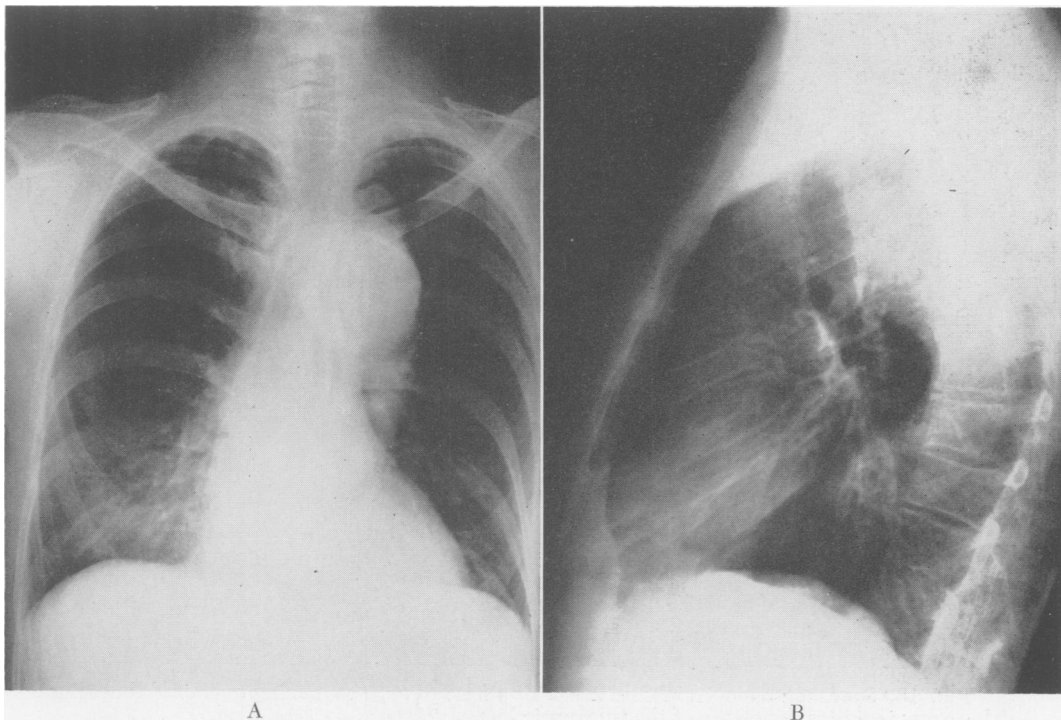
B

FIG. 11 A and B. Roentgenograms of chest in Case 2, in postero-anterior and lateral projections, showing a fusiform aneurysm involving the distal arch and descending thoracic aorta.
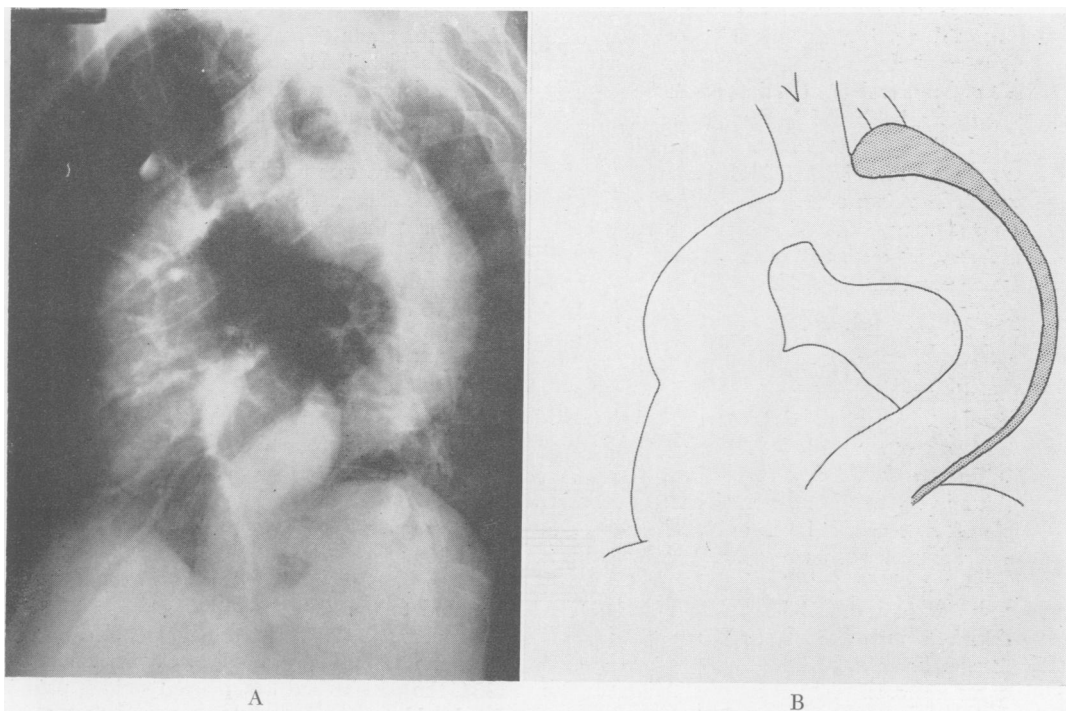

FIG. 12. Angiocardiogram in Case 2, showing outline of dissecting aneurysm arising near the anomolous right subclavian artery and extending to the level of the diaphragm. The true lumen of the arota is more densely opacified than the false passage.
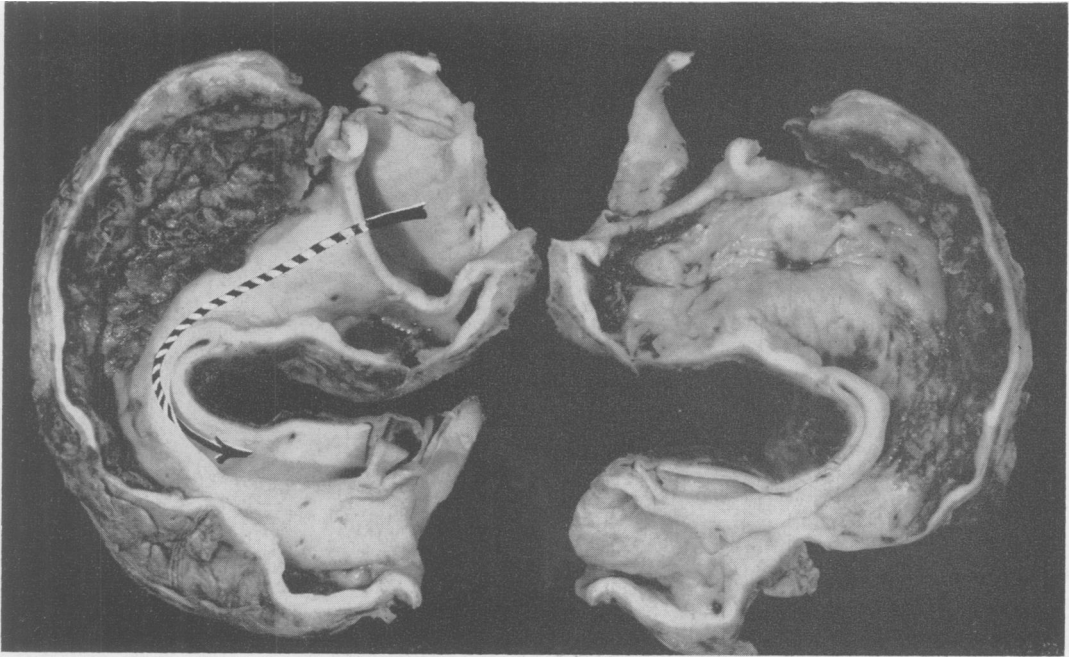
FIG. 13. Specimen removed in Case 2, showing false passage containing organized thrombus. The arrow indicates the course of the true aortic lumen.
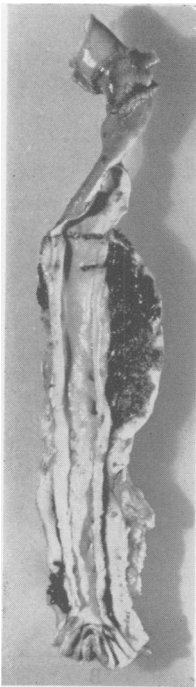

FIG. 14. Autopsy specimen in Case 2, showing intact aortic homograft and extent of the dissecting process into the abdominal aorta and iliac arteries.

midthoracic aorta (Fig. 16). Beyond this point the aorta was dilated to the level of the diaphragm. Occluding clamps were applied proximal to the left subclavian artery and distal to the globular lesion, and the intervening segment was excised. The dissected passage was obliterated below by approximating the outer and inner walls with a continuous suture, and the aortic defect was bridged with a freeze-dried aortic homograft (Fig. 17). The aorta was completely occluded for 34 minutes. The postoperative course was uneventful except for moderate paresthesia in her lower extremities, and the patient was discharged from the hospital 13 days later, in good condition.

Examination of the specimen disclosed that the proximal point of resection was above the original site of intimal perforation and the dissected passage, while distally two lumens were observed extending around the entire circumference of the aorta (Fig. 18).

Case 4. E.H., a 61-year-old Negro man, was admitted to the Veterans Administration Hospital on January 25, 1955, with a diagnosis of dissecting aneurysm of the thoracic aorta. On December 22, 1954, the patient had experienced sudden pain in the shoulder and substernal area, which was severe and resulted in his sudden collapse. On admission to another hospital blood pressure was 200/140
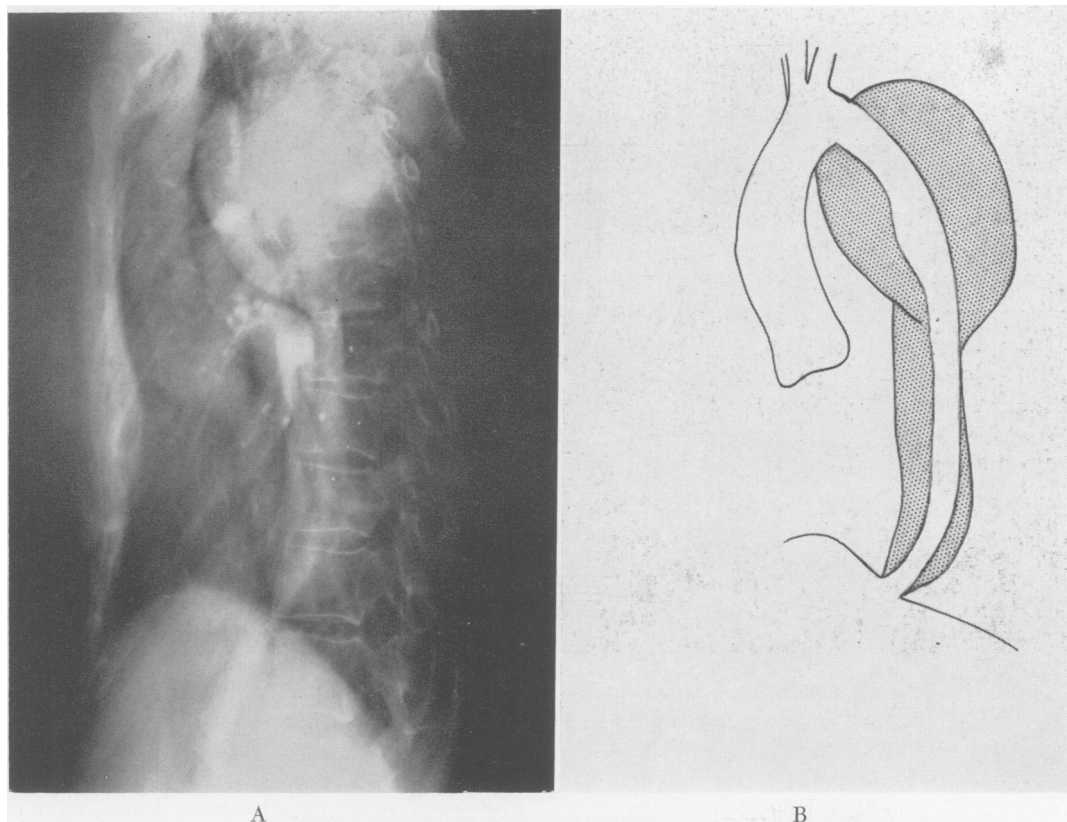
FIG. 15. Angiocardiogram made in Case 3, showing an extensive dissecting aneurysm arising distal to the left subclavian artery.

mm. Hg, and the electrocardiographic tracing and roentgenogram of the chest were found to be normal (Fig. 19). During the next several days the pain gradually subsided, but 6 days after onset of symptoms roentgenograms of the chest revealed the presence of a mass above the aortic knob on the left (Fig. 20).

Physical examination at the Veterans Administration Hospital revealed blood pressure of 170/140 mm. Hg. Prominent pulsations were observed in the suprasternal notch and in the left supraclavicular area. There were no cardiac murmurs, and the remainder of the physical examination was within normal limits.

Laboratory studies revealed a moderate secondary anemia, and a negative serologic test for syphilis.

Roentgenograms of the chest showed a homogenous mass in the left superior mediastinum, which on lateral projection appeared to arise from the posterior portion of the aortic arch (Fig. 21). The aorta was widened in its ascending and descending thoracic portions. Other radiologic procedures, including aortic angiography, laminography, and fluoroscopy, confirmed the diagnosis of aortic aneurysm.

On February 9, 1955, operation was performed through a left posterior-lateral incision with resection of the fifth rib. Preliminary exploration revealed the mass to arise from the distal arch of the aorta, just beyond the left subclavian artery. The mediastinal pleura overlying the aorta was excised, and the aorta proximal and distal to the aneurysm was exposed by dissection. The base of the aneurysm was isolated, and a non-crushing clamp applied at its junction with the aorta. The sac was opened distal to the clamp and found to be filled with a laminated thrombus. The aortic wall was repaired with a row of interrupted mattress sutures, and the cut edges were oversewn with figure-of-eight sutures.

The postoperative course was uneventful, and the patient was discharged on February 24, 1955. Histologic examination of the resected sac revealed a perforation of the aorta had occurred, and a local-
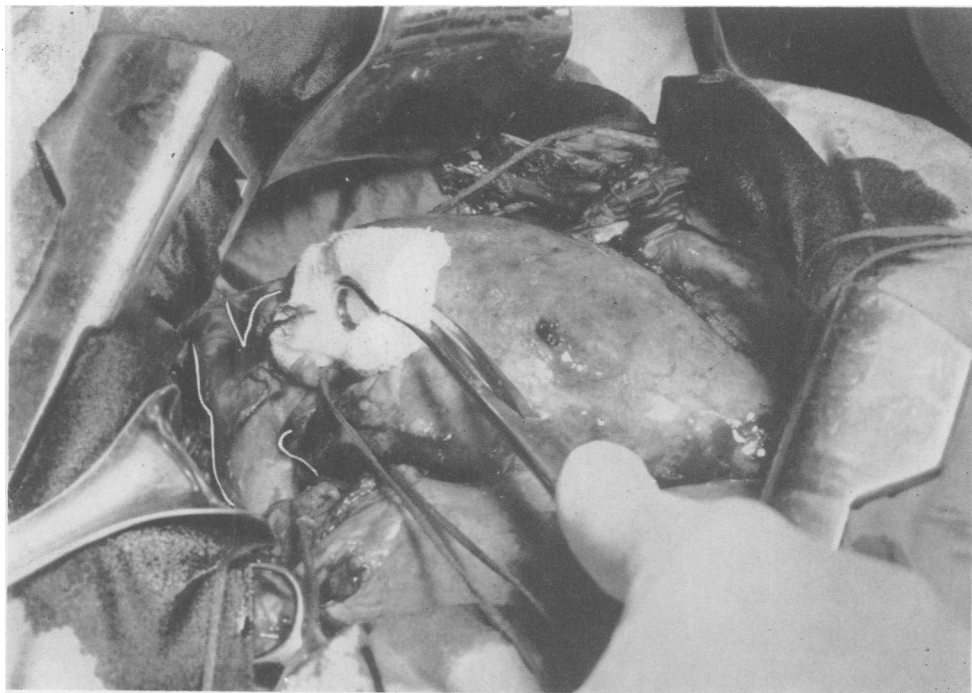
FIG. 16. Photograph made at operation in Case 3, showing globular portion of dissecting aneurysm originating distal to the left subclavian artery.

FIG. 17. Photograph made at operation in Case 3, showing aortic homograft bridging defect after excision of aneurysm.
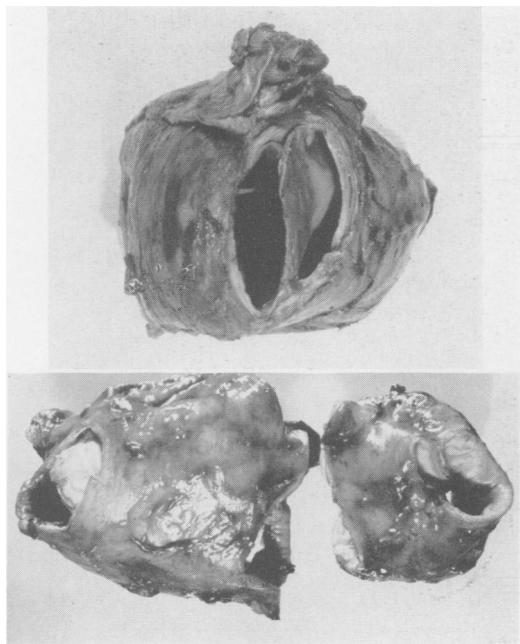
FIG. 18. Photograph of specimen removed in Case 3, showing double lumen created by the dissecting process.
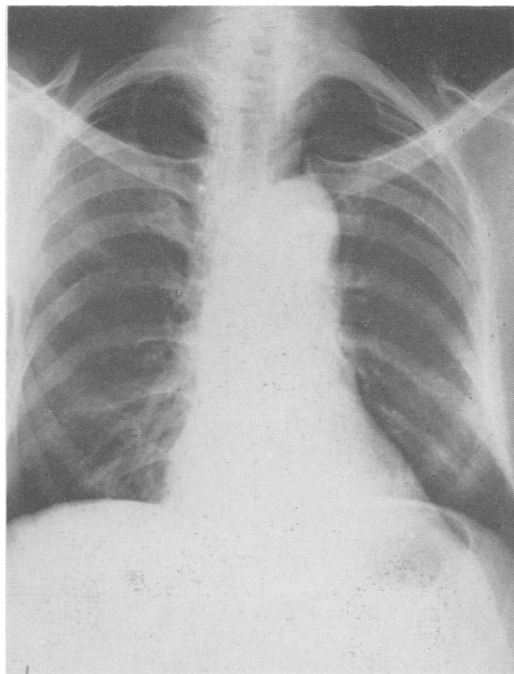

FIG. 19. Roentgenogram of chest in Case 4, made at the time of onset of symptoms, showing moderate prominence of the aortic shadow.

ized area of aorta was involved, with the adventitial layers elevated and containing the aneurysm which lay outside the medial coat.

Case 5. F.P., a 43-year-old white man, was admitted to Methodist Hospital on February 20, 1955, 2 days after the sudden onset of excruciating pain in the lumbar region posteriorly. Soon after the pain began the left thigh became painful, and later the leg became cold and numb. The patient had been known to have hypertension for about 20 years, with the blood pressure ranging around 200/110 mm. Hg responding poorly to drug therapy.

Physical examination upon admission revealed a blood pressure of 160/80 mm. Hg in both upper extremities, 230/110 in the right lower extremity, and no blood pressure was obtainable in the left lower extremity. His pulse was rapid and irregular, and the skin was cool and moist. The heart was enlarged, and there were systolic and diastolic murmurs in the aortic valvular zone. The left lower extremity was cool and pulseless.

Roentgenogram of the chest revealed cardiac enlargement with elongation, broadening, and tortuosity of the thoracic aorta (Fig. 22). The electrocardiogram showed a gross irregularity, consisting of multifocal ectopic beats with short runs of
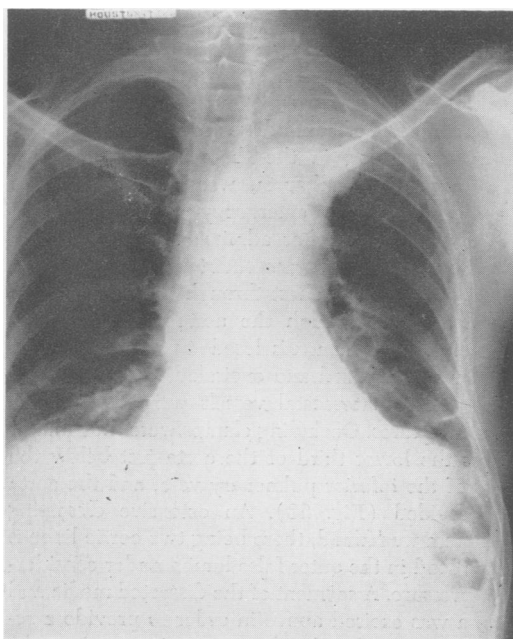

FIG. 20. Roentgenogram of chest in Case 4, made six days after onset of symptoms, showing a mass in the left superior mediastinum confluent with the aortic shadow.
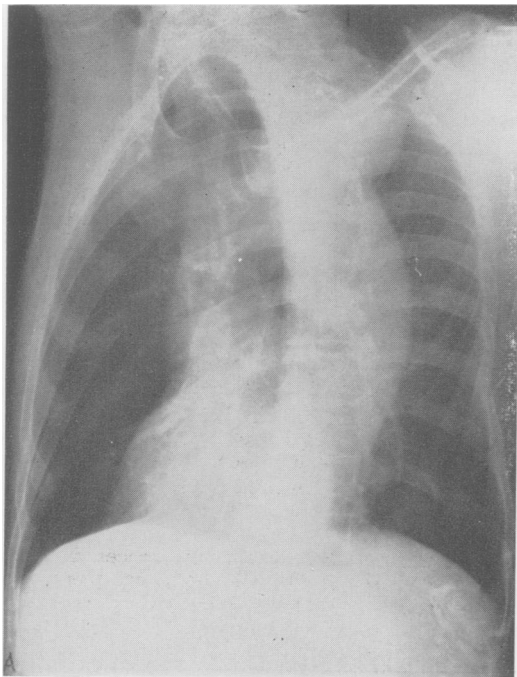
FIG. 21. Roentgenogram of chest in Case 4, in oblique projection, showing aneurysm arising from the distal portion of the aortic arch.

FIG. 22. Roentgenogram of chest in Case 5, showing widening of the supracardiac mediastinal shadow.

auricular flutter and fibrillation. The ST changes were indicative of coronary obstruction or pericarditis. Significant laboratory findings included a moderate leukocytosis, and a blood urea nitrogen of 62 mg. per cent.

A diagnosis of acute dissecting aneurysm of the aorta, originating in the thoracic and extending into the abdominal aorta, was made. Operation was performed as an emergency procedure approximately 2½ hours after admission. Under general anesthesia a left thoraco-abdominal incision was made, entering the chest through the eighth intercostal space. Although the aorta was of normal diameter, palpation disclosed two lumens. The aorta was mobilized and encircled with tapes, and two pairs of intercostal vessels were divided between ligatures. Occluding clamps were then placed across the lower third of the aorta just below the level of the inferior pulmonary vein, and the aorta was divided (Fig. 23). An extensive dissecting aneurysm was found, there being two aortic lumens with blood in the outer false lumen under considerable pressure. A segment of the dissected media and intima was excised above in order to provide a reentry opening for the dissecting passage into the normal lumen (Fig. 23). The layers of the vessel were then sutured to one another with a continuous suture (Fig. 23). In the inferior segment the layers
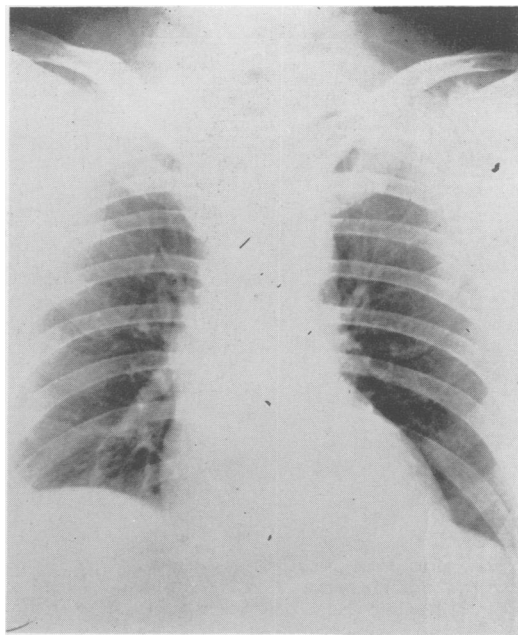
were sutured together around the entire circumference of the aorta. The ends of the aorta were then reanastomosed (Fig. 23). When the clamps were removed after 18 minutes of occlusion, a marked drop in blood pressure ensued, but this responded to the administration of norepinephrine. The abdominal aorta was then exposed retroperitoneally, and encircled with tapes below the level of the inferior mesenteric artery. Clamps were applied and the aorta divided just above the bifurcation. It was apparent that the dissection had extended to this point, and two lumens were again noted. Partially clotted blood in the outer false lumen was aspirated above and below. The dissected wall of the aorta was then resutured to its normal position in the superior and inferior segments, and the aorta was re-anastomosed. Upon completion of the operation the blood pressure was 130/60 and the pulse was irregular.

Circulation in the left leg improved steadily, and 12 hours after operation pedal pulses were palpable on the left as well as on the right side. By the fifth day after operation blood urea nitrogen had dropped to 19 mg. per cent, and urinary output was normal. On the fourth day after operation evidence of aortic insufficiency began to increase, and was accompanied by auricular flutter. Nevertheless, there was gradual improvement in the patient's condition, and by February 27, 1955, a normal sinus rhythm returned, accompanied by im-
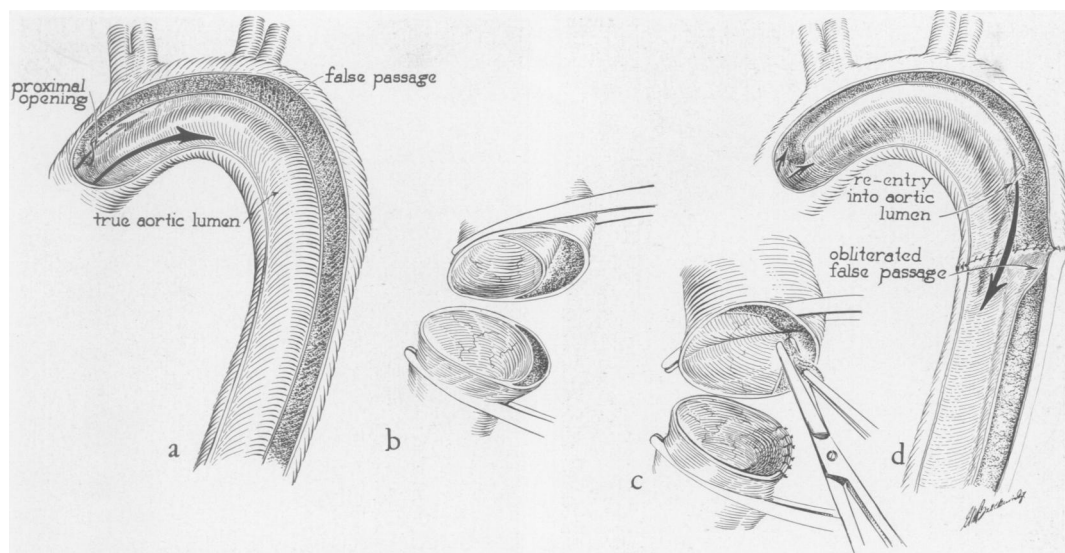
FIG. 23. Drawing showing in (a) the site of origin and extent of the dissecting process in the thoracic aorta in Case 5. In (b) the aorta has been divided. In (c) the false lumen has been obliterated distally, and proximally a segment of the inner layer is being excised to create a re-entry passage. In (d) the anastomosis is completed.

provement in the electrocardiographic pattern. On February 28, eight days after operation, the patient suddenly expired.

Autopsy revealed a massive hemopericardium produced by rupture of the dissecting aneurysm into the pericardial sac. The original perforation of intima and media was extensive, and was located approximately 3 cm. above the coronary ostia. The repairs of the thoracic and abdominal aorta segments were intact and appeared to have functioned well.

Case 6. E.L., a 72-year-old white man, was admitted to the Methodist Hospital on February 27, 1955, 3 weeks after an episode of severe pain in the chest. The pain developed suddenly, and radiated down both arms into the epigastrium, and posteriorly to the lower thoracic and upper lumbar region. He was taken in a state of shock to another hospital, where a blood pressure of 80/40 mm. Hg was obtained. During the ensuing week, the pain gradually subsided, but a roentgenogram of the chest revealed a left pleural effusion. Thoracentesis was performed, and about 300 cc. of bloody fluid was obtained.

On admission to the Methodist Hospital the blood pressure was 200/98 mm. Hg. There was evidence of fluid in the left chest, but otherwise the physical examination was essentially negative.

Routine laboratory studies were not abnormal. Roentgenograms of the chest indicated the presence of pleural fluid on the left, slight cardiac enlargement, and a diffuse widening and elongation of the thoracic aorta (Fig. 24). Roentgenograms made about one year previously disclosed a relatively normal aortic shadow (Fig. 25). Angiocardiography revealed a tortuous distal aortic arch, and a double shadow in the descending thoracic aorta which was suggestive of a dissecting aneurysm (Fig. 26). The electrocardiogram revealed single premature ventricular beats, but it was otherwise within normal limits.

On March 4, 1955, thoracotomy was performed through a left posterolateral incision. About 500 cc. of dark liquid blood was found in the left pleural space. The descending thoracic aorta was tortuous, and constricted in its midportion for a distance of about 2½ cm. Below this point, however, the aorta was enlarged, and by palpation a double lumen was detected. The aorta was mobilized above and below the area of constriction. Occluding clamps were placed across the aorta, and it was transected, revealing a dissecting aneurysm involving the entire circumference of the wall (Fig. 27). Upon release of the distal clamp only slight retrograde blood flow occurred from the false lumen. A window was cut in the intima above, and the remainder of the wall was then sutured to obliterate the false passage. Distally the dissected layers were approximated by a continuous circular suture, and the ends of the aorta were anastomosed (Fig. 28).
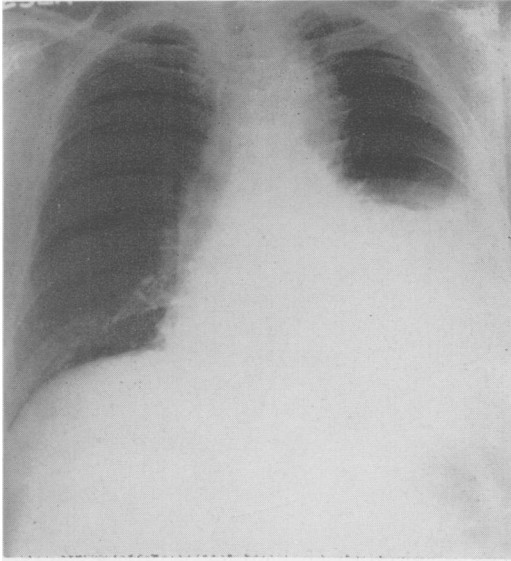
FIG. 24. Roentgenogram of chest in Case 6, showing presence of pleural fluid in left hemithorax and enlargement of the aortic shadow.
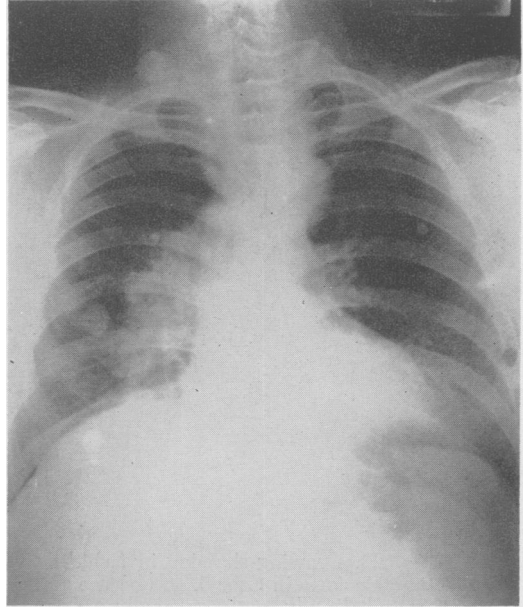

FIG. 25. Roentgenogram of chest in Case 6, made one year previously, showing moderate prominence of the aortic shadow but no evidence of aneurysm formation. A broncho-pneumonic process is demonstrated on the right.

Postoperatively, the patient's course was uneventful, and he left the hospital 18 days later in good condition.

DISCUSSION

Although not a common disease, dissecting aneurysm of the aorta occurs with sufficient frequency to merit clinical interest. Its incidence has been variously reported from a little more than 0.1 to about 1 per cent of necropsies,[2, 5, 10, 16, 28] but there is reason to believe that the actual incidence of the disease may be considerably higher than that generally reported.[19] Males are affected about twice as often as females, with the highest age incidence in the fifth to seventh decades.

The etiology is not clear, although extensive studies have been made of the pathogenesis and pathology of the disease, providing useful information in the consideration of an effective surgical approach to the problem. The underlying predominant lesion appears to be degeneration of the elements of the media, which may be localized or diffuse.[23, 26] The pathogenesis of the initial rupture or tear in the intima

and media is not well understood, but various mechanical factors have been suggested, including conversion of the longitudinal force during diastole into a transverse force, with lateral stretching and distention of the aorta, fixation of the ends of the aortic arch by the pulmonary artery and bronchi, and sudden increase in blood pressure from trauma or other causes. The dissection commonly begins as a transverse tear in the intima and media a few centimeters above the aortic valves, or in the descending thoracic aorta near the origin of the left subclavian artery or in the ligamentum arteriosum. Once this occurs, separation of the intramural layers of the aorta by the forceful stream of blood produces dissection, usually at the junction of the middle and outer thirds of the media, and progresses distally, involving all or a portion of the circumference of the aorta. As branches are encountered, they may be sheared off or the dissecting process may extend along them for varying
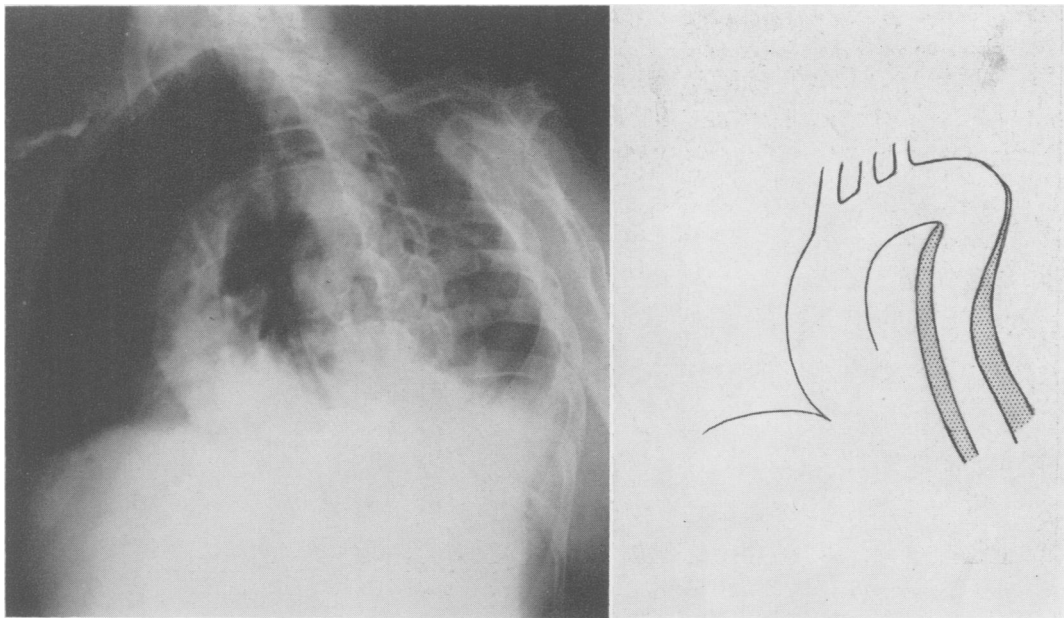
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FIG. 26. Angiocardiogram made before operation in Case 6, showing a tortuous descending thoracic aorta and the presence of a dissecting aneurysm.

distances, thus diminishing or completely interrupting the blood supply to these areas. The extent and course of the dissection is quite variable, as demonstrated by Shennan's studies (Fig. 1). In the most acute and severe form there is rapid dissection and finally terminal perforation through the adventitia into the pericardium, mediastinum, pleura or peritoneal cavities, with death in a few hours or days. In the subacute type, the process may begin abruptly, but then progresses gradually over a period of days or weeks, with terminal adventitial rupture and death. In the chronic form re-entry of the dissected passage into the lumen of the aorta usually takes place, thus forming a "double-barreled" aorta. Under these circumstances the false passage may become covered with endothelium, or rarely be obliterated by thrombus formation with subsequent fibrous tissue organization. This eventuality, i.e., re-entry of the dissected passage, is therefore of considerable importance in the course of the pathologic process, since it provides a means for control and even re-

pair of the process. Perhaps more significant is the fact that it suggests a clue to an effective surgical approach to the problem.

In a number of recent reports emphasis has been placed upon the characteristic clinical manifestations of dissecting aneurysms of the aorta, the awareness of which should lead to early diagnosis.[2, 6, 7, 11, 14] The condition generally develops in patients with a previous history of hypertension. The onset is usually sudden, and is characterized by severe, agonizing pain in the chest or epigastrium, which radiates along the course of the ribs, to the back or down into the abdomen. In some instances the onset is marked by loss of consciousness. Shock with significant lowering of the blood pressure is frequently present. A variety of cerebral or peripheral symptoms may develop as a result of interference with blood supply to the central nervous system or to the extremities. In a series of 26 cases reported by Moersch and Sayre,[18] 46 per cent had neurological complications. In this group cerebral symptoms predominated, but dramatic spinal
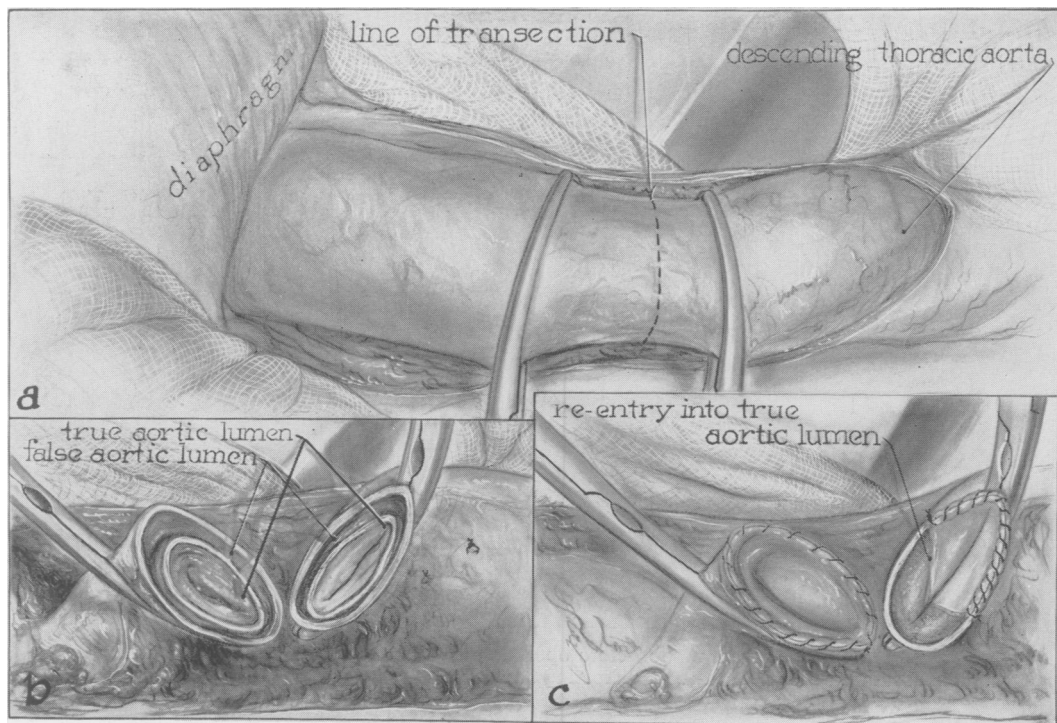
FIG. 27. Drawing made at operation in Case 6, showing in (A) occluding clamps applied to descending thoracic aorta, in (B) the appearance after dividing the aorta of the true and false lumens, and in (C) the method of obliteration of the false lumen distally and creation of a re-entry passage proximally.

cord symptoms also were noted. Interference with peripheral arterial flow in the extremities is also a frequent early manifestation of dissecting aneurysm of the aorta.

The presence of an aortic diastolic murmur is a significant diagnostic feature in from one-fourth to one-half the cases, although the mechanism for its development is not clear. In almost every instance some type of electrocardiographic abnormality can be demonstrated.[14] The most common pattern is one indicative of left ventricular strain. When the diagnosis is in doubt and symptoms are consistent with coronary occlusion, the absence of a pattern of myocardial infarction should suggest the diagnosis of dissecting aneurysm. The presence, however, of an electrocardiographic picture of acute coronary occlusion does not exclude presence of dissecting aneurysm.

Roentgenologic studies are particularly valuable in supporting the diagnosis.[15, 22] The first case of dissecting aneurysm of the aorta in which the clinical diagnosis was confirmed by this means was reported by Davy and Gates[3] in 1922. The chest roentgenogram was reported as showing widening and radiolucence of the aortic arch and descending thoracic aorta. The most common finding is widening of the supracardiac shadow, involving the entire thoracic aorta, or only a small segment of it (Fig. 22). Roentgenograms taken prior to onset of dissection are valuable for evaluating the extent of increase in size of the thoracic aorta (Figs. 19, 20, 24, 25). In some instances extravasation of the blood into the mediastinum will accentuate the aortic shadow. Dissection along one of the major branches of the aorta may also be demonstrated roentgenographically, and is
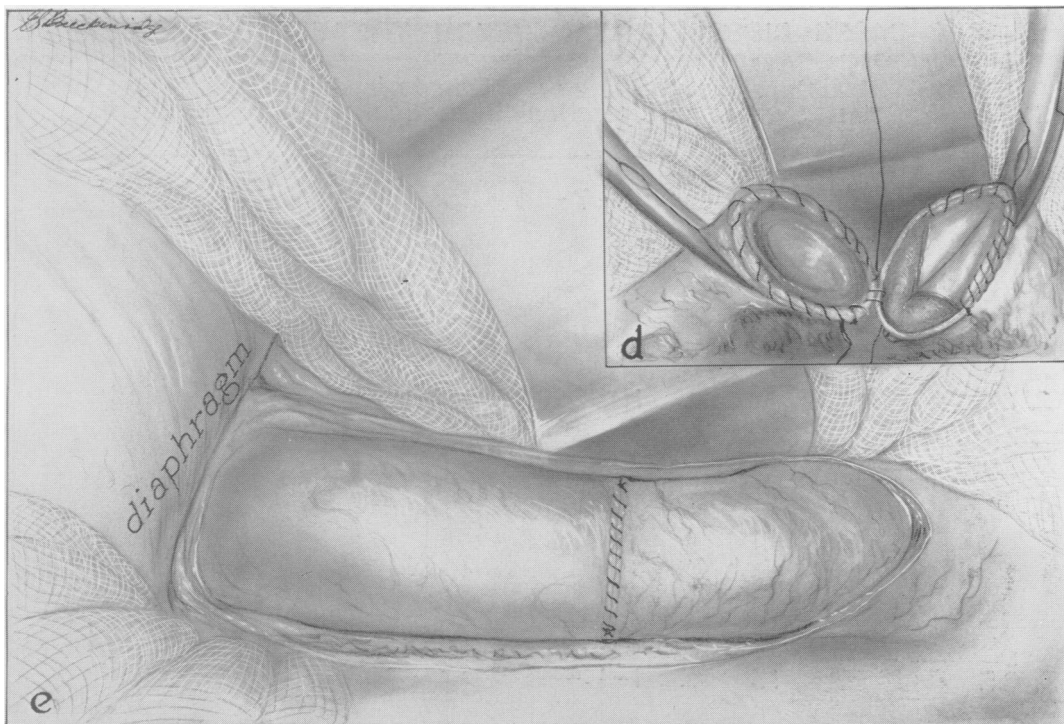
Fig. 28. Drawing made at operation in Case 6, showing in (D) the method of anastomosis of the repaired ends of aorta, and in (E) the completed anastomosis.

considered an important diagnostic feature.[30] The formation of a hematoma within the wall of the aorta sometimes produces a marked thickening of the wall which is visible on the roentgenogram, the false passage appearing more radiolucent than the dense true lumen of the aorta. When intimal calcification has formed along the endothelial surface of the false passage, the aorta may have a double-barreled appearance.

The advantages of sectional roentgenography have been emphasized by Scott and Bottom,[24] who feel that in any case in which diagnosis is not certain from the clinical manifestations and routine chest roentgenograms, sectional laminography should be employed. By this method widening of the ascending aorta and arch can be clearly demonstrated, and non-vascular mediastinal lesions may be separated from the aorta.

The most valuable method of diagnosing dissecting aneurysm of the aorta

roentgenographically is that of angiocardiography. This procedure was first employed by Paullin and James,[20] and by Golden and Weems,[8] whose reports were published separately but concerned the same case. Characteristically in this type of roentgenogram the dye is heavily concentrated in the true aortic lumen, while the false passage appears as a lateral or medial, somewhat superimposed, less dense channel. By utilizing postero-anterior, lateral, or oblique positions, almost any dissecting aneurysm of the thoracic aorta should be demonstrated by this method. These characteristic features are well illustrated in our cases (Cases 2, 3; Figs. 12, 15, 26).

As indicated above, the treatment of dissecting aneurysms of the aorta has been almost entirely symptomatic, and the few efforts to attack the problem surgically have all proved unsuccessful. An analysis of the clinical and pathologic features of the disease, however, would suggest that

SIX CASES DISSECTING ANEURYSM TREATED SURGICALLY

Case	Blood Pressure	Duration of Symptoms	Date of Operation	Extent of Dissection	Type of Operation	Results
1. P.M. 58 w.m.	$\frac{230}{130}$	12 mos.	7-7-54			good
2. W.C. 46 w.m.	$\frac{170}{112}$	6 mos.	11-23-54			died
3. C.F. 40 w.f.	$\frac{115}{80}$	13 mos.	1-10-55			good
4. E.H. 61 c.m.	$\frac{200}{140}$	1 mo.	2-9-55			good
5. F.P. 43 w.m.	$\frac{200}{110}$	2 days	2-20-55			died
6. E.L. 72 w.m.	$\frac{200}{98}$	1 mo.	3-4-55			good

TABLE I.

surgical treatment may be feasible and effective. Depending upon the nature of the lesion, whether localized or diffuse, surgical therapy may be directed toward correction of the process by actual removal and repair of interruption of its progress, and the creation of conditions compatible with function and eventual repair. Accordingly the problem is concerned essentially with the prevention of further intramural dissection and terminal rupture through the outer adventitial layer and restoration of blood flow through the normal lumen. The rationale of the procedure is based upon nature's method of healing, in which spontaneous re-entry of the dissected passage at some distal point in the aorta permits restoration of peripheral circulation, and removes the increasing tension upon the outer wall, thus preventing its rupture. This may then lead to the formation of a double-barreled aorta from the origin of the dissection to the point of re-entry, and as a consequence of equalizing the pressures in the true and false lumens sufficient periaortic fibrous tissue response occurs to strengthen the outer wall and prevent rupture. Under some circumstances apparently this eventually results in sufficient diminution of pressure in the false passage to permit thrombosis and obliteration by fibrous organization. To be sure in the natural course of the disease these events are relatively uncommon, occurring perhaps in less than one fourth or one fifth of the cases. The fact remains, however, that their occurrence provides virtually the only means of recovery, and they are compatible with survival.

Fortunately in most cases, and even in the acute forms of the disease, terminal rupture and death may be delayed for hours or days. Sufficient time is, therefore, available to permit application of surgical therapy. In such instances, as illustrated in our Case 5, operation should be considered an emergency procedure.

Although the essential principles underlying the operative procedure are the same for most cases, one of several surgical approaches may be employed, depending upon the nature of the lesion. In cases, for example, in which the dissection begins in the ascending or descending part of the aortic arch, the most logical procedure would seem to be the creation of a re-entry passage into the aortic lumen above, with obliteration of the false passage below (Case 6). This may be accomplished by cross-clamping the descending thoracic aorta, dividing it completely between clamps, obliterating the false passage below by approximating the outer and inner layers, excising a small segment from the inner intimal and medial layer above to produce re-entry of the outer lumen, and then completing the procedure by end-to-end anastomosis (Figs. 23, 27 and 28; Cases 5 and 6). Thus, blood flow from the double aortic lumen above is diverted into the single normal lumen below. Prior to approximating the outer and inner layers in the distal end of the divided aorta, the occluding clamp should be momentarily released to permit escape of the blood and clots in the false passage. Suction may also be used to help empty its contents. In some instances, as in our Case 5, where the dissection has extended into the terminal portions of the aorta and iliac vessels, it may be desirable to repeat the above procedure in the abdominal aorta just above the bifurcation in order to effect better emptying of the false passage below, and thus provide prompt release of its compressive effect on the normal aortic lumen.

It is important, however, in our opinion, to apply this procedure primarily to the descending thoracic aorta rather than peripherally, although the latter may be done as an additional procedure. Re-entry of the false passage into the normal lumen as close to the point of origin of the dis-

secting process as may be feasible would seem desirable for a number of reasons. For one thing, by shortening the extent of the false passage there is a tendency to reduce peripheral resistance to blood flow, and thus diminish the likelihood of rupture of the weakened outer wall until it can be strengthened by periaortic fibrous tissue response. For another, restoration of blood flow through the normal aortic lumen in the thoracic aorta and above the abdominal aorta tends to correct the compressive ischemic effects of the false passage upon the points of origin of the major vascular channels of the abdominal aorta. Accordingly, this permits more effective circulation through these major visceral channels. In this connection it may be significant that in both of the cases reported by Gurin, Bulmer and Derby,[9] and by Shaw,[25] in which a re-entry point was created in the terminal portions of the aorta, death occurred about a week later from renal insufficiency.

Another factor which we believe is important in the management and success of the operative procedure is control of hypertension during both the operation and the postoperative course. This is well illustrated by our Case 5. This patient, who had long had severe hypertension, showed progressive improvement both in return of circulation in the lower extremity and in renal function during the first week following emergency operation. Unfortunately, in spite of efforts to maintain blood pressure at lower levels, it continued to rise steadily, and with resumption of his severe hypertension the weakened outer wall gave way, resulting in death.

The principles of the operative procedure described above may be combined with excisional therapy under certain circumstances where the lesion is fairly well localized and arises at or below the level of the left subclavian artery, thus permitting a more curative type of procedure. This may be done by excision of the seg-

ment involved in the origin of the dissection, followed by end-to-end anastomosis after obliterating the distal false passage, as illustrated by our Case 1 (Figs. 3–8). In other instances the resulting defect may require the use of an aortic homograft, as illustrated in our Cases 2 and 3 (Fig. 17).

Still another but probably uncommon approach to the problem is that of excision with repair by aortorrhaphy, as illustrated by our Case 4. Here the dissection, while relatively acute, was unusually limited and well localized, and it was therefore possible to excise it completely and repair the defect by suture approximation of the lateral margins of the aortic wall.

There were two deaths among our six cases (Table I). In one this was due to rupture of the outer wall of the dissecting aneurysm at one of the most common sites for this terminal event, namely into the pericardium. As indicated above it is believed that this resulted from inability to control resumption of hypertension. The other death was due to the consequences of ventricular fibrillation precipitated by tension pneumothorax and hypothermia. In this connection hypothermia was employed to counteract the ischemic effects of temporary arrest of aortic circulation in two cases. It may be significant that neither of these patients, nor any of the seven other cases of fusiform aneurysms of the thoracic aorta treated by resection with homograft replacement in whom hypothermia was used, showed any ischemic manifestations of temporary arrest of aortic circulation up to approximately one hour.[1, 4] On the other hand, in one of the cases reported above (Case 3) and in three of four other cases of fusiform aneurysms of the aorta treated by resections and graft replacement in which operation was done without hypothermia, some manifestations of ischemic damage to the spinal cord occurred. Fortunately, all but one in the latter group recovered completely. This experience, however, suggests that hypothermia is a

useful procedure for this purpose in cases in which temporary occlusion of the aorta is necessary at or proximal to the level of the sixth or seventh thoracic vertebra.

The results obtained in the four patients who recovered from the operation have been quite gratifying. All are completely free of symptoms, and have resumed normal activity. Better evaluation of this method of operative treatment of dissecting aneurysms obviously requires further experience and longer follow-up observations. On the basis, however, of these early gratifying results there is reason to believe that this method of therapy offers a more effective approach to the problem than has been previously available.

SUMMARY

1. Dissecting aneurysms of the aorta are associated with grave disturbances and a rapidly fatal course in from 75 to 90 per cent of the cases.

2. The etiology has not been determined precisely, but studies of the pathogenesis and pathology suggest that the underlying predominant lesion is degeneration of the elements of the media. The dissection commonly begins as a transverse tear in the intima and media a few centimeters above the aortic valves or in the descending thoracic aorta near the origin of the left subclavian artery. Once this occurs, separation of the intramural layers of the aorta by the forceful stream of blood produces dissection, usually at the junction of the middle and outer thirds of the media, and progresses distally, involving all or a portion of the circumference of the aorta.

3. While the extent and course of dissection vary, in general, three patterns of the disease may be recognized. In the most acute and severe form there is rapid dissection and terminal perforation through the adventitia, with death in a few hours or days. In the subacute type the process may begin abruptly, but then progresses gradually over a period of days or weeks with terminal rupture and death. In the chronic form, re-entry of the dissected passage into the lumen of the aorta usually takes place, thus forming a double-barreled aorta.

4. Increasing awareness of the problem, and recognition of the characteristic clinical manifestations, should lead to more frequent early diagnosis. Roentgenologic studies are particularly valuable in supporting the diagnosis, and it often can be confirmed by angiocardiography, which characteristically reveals a double shadow representing the true and false aortic lumens.

5. Treatment has been almost entirely symptomatic and unsatisfactory, and the few previous efforts to attack the problem surgically have all proved unsuccessful.

6. The rationale of the surgical procedures proposed in this report is based upon nature's method of healing, in which spontaneous re-entry of the dissected passage at some distal point in the aorta permits restoration of peripheral circulation and removes the increasing tension upon the outer wall, thus preventing its rupture. This may be achieved by one of several surgical approaches utilizing essentially similar underlying principles. In cases in which the dissection begins in the ascending or descending part of the aortic arch, the most logical procedure would seem to be the creation of a re-entry passage into the aortic lumen above, with obliteration of the false passage below. This is achieved by cross-clamping the descending thoracic aorta, dividing it completely between clamps, obliterating the false passage below by approximating the outer and inner layers, excising a small segment from the inner intimal and medial layer above to produce re-entry of the outer lumen, and then completing the procedure by end-to-end anastomosis. In other cases in which the lesion is fairly well localized or arises at or below the level of the left subclavian artery, this procedure may be combined with excision of the segment involved in the origin of the dissection followed by

end-to-end anastomosis or insertion of an aortic homograft.

7. Six cases are reported in which these methods of surgical approach were employed with two deaths. One occurred on the 8th postoperative day from rupture of the outer wall of the dissecting aneurysm, presumably resulting from inability to control hypertension. The other was due to the consequences of ventricular fibrillation precipitated by tension pneumothorax and hypothermia. In the remaining four patients recovery was uneventful, and the results have been gratifying.

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